Developmental relations between ADHD symptoms and reactive versus proactive aggression across childhood and adolescence

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Abstract: OBJECTIVE: Past research has provided some preliminary evidence that ADHD and reactive aggression have overlapping neurocognitive bases. Based on this, we tested the hypothesis that ADHD symptoms are closely coupled in developmental terms with reactive aggression, more so than with proactive aggression with which it has been postulated to be only indirectly linked. METHOD: We used latent growth curve analysis to estimate the developmental relations between ADHD symptoms and subtypes of aggressive behavior in a normative sample of 1,571 youth (761 female, 810 male) measured from ages 7 to 15. RESULTS: Individual ADHD trajectories were significantly and substantially correlated with individual trajectories in both aggressive subtypes; however, consistent with our hypothesis, the relation with reactive aggression was significantly stronger. CONCLUSION: Our study provides some of the first evidence for a differential relation between ADHD symptoms and aggression subtypes not only cross-sectionally but also in terms of their longitudinal developmental trajectories.

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Developmental relations between attention-deficit/hyperactivity disorder (ADHD) symptoms and reactive versus proactive aggression

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Abstract

Past research has provided some preliminary evidence that ADHD and reactive aggression have overlapping neurocognitive bases. Based on this, we hypothesised that ADHD symptoms would be closely coupled in developmental terms with reactive aggression; more so than with proactive aggression with which it has been postulated to be only indirectly linked. We used latent growth curve analysis to estimate the developmental relations between attention-deficit/hyperactivity disorder (ADHD) symptoms and subtypes of aggressive behaviour in a normative sample of 1571 youth (761 female, 810 male) measured from ages 7 to 15. Our results suggested that individual ADHD trajectories were significantly and substantially correlated with individual trajectories in both aggressive subtypes; however, consistent with our hypothesis, the relation with reactive aggression was significantly stronger. This provides some of the first evidence for a differential relation between ADHD symptoms and aggression subtypes not only cross-sectionally but also in terms of their longitudinal developmental trajectories.

Attention-deficit hyperactivity disorder (ADHD) is, at the clinical level, defined by impairing levels of inattention and hyperactivity (APA, 2013; Barkley, 1997; Willoughby, 2003). An important correlate of ADHD symptoms is aggressive behaviour. Aggression commonly presents in clinical ADHD, where it is a major source of impairment and often represents the trigger for initial referral for diagnostic assessment (Jensen et al., 2007; King & Waschbush, 2010). In the long term, aggressive behaviour associated with ADHD has important consequences for the functioning of an individual within their social environment and likely contributes to the adverse outcomes associated with ADHD through, for example, increasing the risk of peer rejection (e.g., Evans et al., 2015; Jester et al., 2005; Jester et al., 2008).

In spite of their established association, only a small number of studies have examined the developmental relations between ADHD symptoms and aggression with a view to illuminating the extent to which they follow correlated developmental trajectories (e.g. Jester et al., 2005; Jester et al., 2008). However, even these studies have tended to focus on general aggression and in posing questions about developmental relations between aggression and ADHD symptoms, a distinction should be made between reactive aggression and proactive aggression. Reactive aggression refers to an impulsive, emotionally 'hot' response to perceived threat or provocation. Proactive aggression, in contrast, refers to premeditated , emotionally 'cool', instrumental behaviours where harm is inflicted intentionally and for the purpose of achieving some end (Kempes, Matthys, De Vries, & Van Engeland, 2005). Aggressive behaviours serving these two functions tend to separate out in factor analyses, show differential patterns of development and relations to other behaviours and outcomes, and respond differently to interventions (e.g., see Babcock, Tharp, Sharp, Heppner, & Stanford, 2014; Fite, Colder, Lochman, & Wells, 2008; Hubbard, McAuliffe, Morrow, & Romano, 2010; Murray, Eisner, & Ribeaud, 2016; Raine et al., 2006). In the current sample, for example, [BLINDED] used growth mixture modelling to explore developmental trajectories of proactive and reactive aggression and found that the former was best characterised in terms of three latent trajectory classes while the latter was best characterised by

four. In other studies, proactive aggression has been shown to be more strongly related to endorsing aggression as an acceptable and productive means of achieving some end while reactive aggression has been shown to be more strongly related to hostile attribution biases, anxiety and peer rejection (Marsee et al., 2008; Raine et al., 2006; Vitaro et al., 2002). In terms of treatment effects, reactive aggression appears to be more responsive to psychosocial and pharmacological interventions than proactive aggression (Barker et al., 2010; Saylor et al., 2016).

The distinction between these subtypes of aggression is important with respect to their potential developmental relations to ADHD. In particular, it seems likely that reactive aggression and ADHD symptoms have a common basis in impaired neurocognitive functions mediating impulse control in the emotion regulation domain (Bennett, Pitale, Vora, & Rheingold, 2004; Saylor & Amman 2016). Core features of both ADHD and reactive aggression appear to be emotional impulsivity i.e. difficulties in inhibiting strong emotional reactions, and emotion dysregulation i.e. the inability to effectively regulate emotional states (e.g., see Saylor & Amman 2016). In contrast, proactive aggression - if associated with ADHD at all – tends to be presumed to be related only indirectly through, for example, peer deviancy training (e.g., Bennett et al., 2004). If this is correct, reactive aggression would be expected to follow a developmental trajectory that is strongly correlated with ADHD symptoms; a trajectory that reflects developmental changes in the underlying common neurocognitive architecture. On the other hand, proactive aggression would be expected to be more weakly related with ADHD in developmental terms. Thus far, there has been only indirect cross-sectional evidence relating to this hypothesis but it is broadly in support of the idea: empirical associations support a strong link between ADHD and reactive aggression but provide weaker and less consistent evidence for a link with proactive aggression (e.g., Bennett et al., 2004; Card & Little, 2006; Dodge, Lochman, Harnish, Bates, & Pettit, 1997; King & Waschbusch, 2010; Retz & Rösler, 2009; Vitaro, Brendgen, & Tremblay, 2002; Waschbusch, Willoughby, & Pelham, 1998). In this study, we use latent growth curve analysis to provide the

first direct test of the hypothesis that ADHD symptoms are developmentally more closely coupled with reactive than proactive aggression.

We focus on a community sample rather than a clinically ascertained sample because while clinical or high risk samples may be better positioned to identify and characterise 'pathological' trajectories, they are not population representative and may be subject to difficulties such as range restriction on the one hand or Berkson's bias on the other. Range restriction is when there is an underestimation of symptom correlations because of a focus on the upper extreme of symptom distributions (Murray, McKenzie, Kuenssberg, & O'Donnell, 2014). Berkson's bias refers to the possibility of overestimating symptom correlations because different symptoms and disorders may independently influence treatment-seeking (Berkson, 1946). This can lead to individuals with multi-morbidity being over-represented in clinical samples (e.g., Maric, Myin-Germeys, Delespual, de Graaf, Vollenbergh & Van Os, 2004). Given the evidence that ADHD symptoms appear to be continuously distributed at the etiological and phenotypic level in the population (e.g., Groen-Blockhuis et al., 2014; Lubke, Hudziak, Derks, van Bijsterveldt, & Boomsma, 2009), it is important to ensure that research does not focus exclusively on clinically ascertained samples. For the same reason, using dimensional measures of ADHD symptoms rather than dichotomous diagnostic status (clinical diagnosis of ADHD versus none) provides a more nuanced and arguably more accurate picture of how symptoms and correlated features of ADHD develop over time.

In utilising growth curve analysis, we model individual trajectories as varying continuously in the population and evaluate the extent to which variations in trajectories for one phenotype (ADHD symptoms) are related to another (proactive or reactive aggression). This kind of analysis provides a useful alternative to growth mixture analyses (e.g. Nagin, 2009) which treat variations in trajectories as categorical and aim to summarise developmental trajectories in terms of a small number of trajectory classes (e.g. see Arnold et al., 2014; Fite, Colder, Lochman & Wells, 2008; Robbers et al., 2011 for examples in ADHD and aggression). The two approaches are complementary, providing different but compatible information about developmental trajectories;

however, with respect to the current study, bivariate growth curve analysis provides a more direct operationalisation of the hypothesis that ADHD symptoms and reactive aggression are closely coupled developmentally in allowing correlations between the components of growth (e.g., intercepts and linear and quadratic slopes) to be estimated.

Method

Participants

The participants were recruited to the BLINDED PROJECT NAME: a longitudinal cohort study concerned with the development of pro- and anti-social behaviours. The sample comprises 1571 children (from a target sample of 1675) who entered one of 56 primary schools in 2004 in (BLINDED COUNTRY/LOCATION)(BLINDED REFERENCES). These schools were selected according to a stratified random sampling procedure that considered school size and location. Compared with those who declined to participate at baseline, the participating sample slightly under-represented children whose parents did not speak German (the official language of the study location) as a first language but were otherwise similar. Data were collected across 8 measurement waves when the children were of median age 7.45, 8.23, 9.21, 10.70, 11.60, 12.63, 13.88 and 15.68. The number of participants contributing data in the current study at each of these waves were 1338, 1314, 1287, 1262, 1061, 972, 1239, and 1267, respectively. Active written parental consent was required for the first six years of participation in the study. To maximise participation parents were offered a financial incentive equivalent to approximately 30 USD. In year 7 of the study (age 13), given BLINDED COUNTRY/LOCATION regulations, the participating youth were required to give their active consent to participate and their parents received an information letter that allowed them to proscribe their child's participation (passive consent procedure; for further details refer to the relevant literature, BLINDED REFERENCES). Youth were offered a financial incentive worth approximately 30 USD for their participation at age 13 and 50 USD at age 15.

Of the 1571 youth contributing data in this study, 870 were male and 805 were female. These youth did not differ significantly on gender [χ^2 (1)=1.23, p=.27] to those in the target sample

who declined to participate. In being based in BLINDED COUNTRY/LOCATION, the sample is diverse in terms of ethnic and cultural background, as indicated by the country of birth and the mother tongue. For example, less than half of the female primary caregivers of the target (36.2%) were born in Switzerland and were German speaking; 6.2% were born in Switzerland but spoke another first language; 6.1% were of Albanian mother tongue (born in former YU or Albania); 8.6% were from former Yugoslavia (other languages); 2.4% were born in Italy; 3.9% were born in Sri Lanka (Tamil language); 3.9% were born in Turkey; 4.8% were born in Portugal; 1.6% were born in Spain; 5.7% were born in Germany; 4% were born in other Western countries; 2% were born in other South/East European countries; 2.6% were born in North Africa or the middle East; 2.4% were born in Sub-Saharan Africa; 4.8% were born in the Far East and 4.8% were born in Latin America.

In terms of socioeconomic status, mean International Socio-Economic Index of Occupational Status (ISEI) score was 44.58 (SD=17.81). This information was not available for the remaining participants. There were too few non-participating individuals with ISEI data to statistically test whether the study sample differed from those not participating on socioeconomic status. Further information on study recruitment, retention and assessment procedures and more detailed descriptions of the sample can be found in previous publications (BLINDED REFERENCES).

Raters

Teachers provided ratings for the current study. Children usually had the same teachers between grades 1 to 3 (ages 7, 8 and 9) and between grades 4 to 6 (ages 10, 11, 12). After this they entered secondary school (ages 13 and 15). Teachers were not compensated for their participation in the first three waves of data but for the remaining waves those with at least seven participants in their class received a book voucher worth approximately 50 USD as incentive to participate. The numbers of teachers providing ratings at measurement waves 1 to 8 were: 113, 148, 217, 274, 265, 258, 366, and 423, respectively.

Measures

Attention deficit symptoms, hyperactivity/impulsivity symptoms proactive aggression and reactive aggression were measured using Social Behaviour Questionnaire (SBQ; Tremblay et al., 1991), completed by teachers. Physical aggression was also measured but not included in the current study because the items could not be clearly classified as referring to either the reactive or proactive aggression behaviours with which our hypothesis was concerned. The items were administered in German and respondents instructed to respond on a five-point Likert scale from (translates to) *never* to *very often*. English-language versions of the items (on which the versions administered in the current study are based) are provided in Table 1.

Previous research has supported the reliability and validity of the SBQ, including as applied to the current sample (BLINDED REFERENCE; Tremblay et al., 1991; Tremblay et al., 1992). The subscales measuring these constructs comprised four items, except reactive aggression which comprised three. Cronbach's alpha for all subscales was a minimum of .86 and mostly >.90. Specifically, for the attention deficit symptom subscales from waves 1-8 Cronbach's alphas were: .94, .95, .95, .95, .95, .95, .95, .94; for hyperactivity/impulsivity they were: .92, .93, .92, .92, .92, .92, .93, .92; for proactive aggression they were .86, .88, .87, .89, .89, .90, .89, .86; and for reactive aggression they were .92, .94, .93, .94, .92, .92, .92, .91.

Statistical Procedure

Overview

We used a latent growth curve analysis (e.g., Curran et al., 2010) to model changes in attention deficit symptoms, hyperactivity/impulsivity symptoms, reactive aggression and proactive aggression over development as well as the correlation between individual trajectories on these phenotypes. Given the complexity of analyses, we used a two-step approach in which factor scores were estimated from latent measurement models for the phenotypes in a first step and factor scores

used in latent growth curve modelling in a second step. We began by fitting univariate growth curve models to each phenotype separately, testing both linear and quadratic growth. We then fit bivariate growth curves to explore pairwise relations between ADHD symptom trajectories and aggression subtype trajectories. All analyses were conducted in *Mplus 7.31* using maximum likelihood estimation (Muthén & Muthén, 2014). Note that this also gives unbiased parameter estimates assuming data are missing at random (MAR).

Measurement models

To obtain factor scores for attention deficit and hyperactivity/impulsivity we used a first-order oblique factor model. This was based on past research and preliminary analyses suggesting that the ADHD items measured correlated but distinguishable factors (e.g., BLINDED REFERENCE). Scaling and identification were achieved by fixing the mean and variance of the latent factors at baseline to 0 and 1 respectively and fixing the intercept and loading of the first item of each first-order factor equal across time. Residual correlations between the same items measured over time were freely estimated. An analogous first-order oblique factor model was fit for proactive and reactive aggression based on the similar considerations. We did not model clustering within teachers at this stage because the children experienced several teacher changes and associated shuffling of clusters and past research in the sample has suggested that the clustering makes only a very small difference to results (BLINDED REFERENCE). Factor score determinacies were examined to ensure the quality of factor scores as proxies for the relevant latent variables. These estimate the correlation between factor scores and the underlying latent variable and are ideally >.90 (Gorsuch, 1983).

Univariate growth curves

We began by fitting linear and quadratic growth curves for each of the phenotypes individually. In this model, the eight observed measures representing the phenotype measured across time were indicators of latent intercept and slope factors. The intercept factor was specified

by fixing its loadings on all eight observed measures to 1. A linear slope factor was specified by fixing its loadings on the eight measures to 0, 0.09, 0.21, 0.39, 0.50, 0.63, 0.78 and 1; reflecting the distance between measurement occasions and a quadratic factor was specified by fixing its loadings to the square of these numbers. Residual covariances between indicators assessed by the same rater across time were also freely estimated. In order to test for quadratic growth, we compared the fits of a model with and without the mean, variance of the quadratic growth factor and its covariances with the intercept and slope factors fixed to zero. In particular, we judged a model including quadratic growth to be superior to one without when the BIC difference favoured this model by more than 10 (Raftery, 1995).

Multivariate growth curves

Two multivariate growth curves were fit: attention deficit symptoms with proactive aggression and reactive aggression and hyperactivity/impulsivity with reactive and proactive aggression. These are simple extensions to univariate growth curves in which individual growth curves are specified as described above for the three phenotypes and the slope and intercept factors allowed to correlate across phenotypes. In addition, residual correlations between different phenotypes measured at the same time were freely estimated to account for excess covariance within compared to between waves due to the same rater being used within but not necessarily across time. In each model, we examined the cross-phenotype correlations in intercepts and linear and quadratic (where applicable) slope parameters. High correlations were interpreted as strong developmental coupling of phenotypes. Unless all phenotypes in the multivariate model showed evidence of both linear and quadratic growth (as opposed to linear growth only), we included only linear growth factors for each phenotype. This decision was taken in order to make the growth correlations as comparable as possible across phenotypes. We tested for differential developmental relations between ADHD symptoms and reactive versus proactive aggression by using a χ^2 difference test in nested model comparison. In the first model, all covariances of the growth

components of reactive and proactive aggression with attention deficit (or hyperactivity/impulsivity) except the those we were of focal interest in our hypothesis test were constrained to equality across phenotypes. That is, the intercept variances of proactive and reactive aggression were fixed equal; the slope variances of reactive and proactive aggression were fixed equal; and slope-intercept covariances between reactive aggression and attention deficit (or hyperactivity/impulsivity) were fixed equal to the corresponding slope-intercept covariance for attention deficit (or hyperactivity/impulsivity) and proactive aggression. The purpose of this first model is to make the covariances in the second model directly comparable. In the second model, additional equality constraints were added: 1) on the attention deficit (or hyperactivity/impulsivity) and reactive aggression versus proactive aggression intercept covariances and 2) on the attention deficit (or hyperactivity/impulsivity) and reactive aggression versus proactive aggression slope covariances. That is, in the second model, the covariance between ADHD and reactive aggression trajectories and the covariance between ADHD and proactive trajectories were constrained to equality. If there was a significant deterioration in fit with the addition of these constraints, this was taken as evidence of differential strength of developmental coupling between ADHD and reactive versus proactive aggression.

Results

Measurement models

The longitudinal oblique first-order factor model for ADHD fit well by conventional criteria (CFI=.97, TLI=.96, RMSEA=.04, SRMR=.04) with within-wave factor correlations ranging from r=.71 to r=.74. The minimum pairwise covariance coverage for items (the proportion of data present for both items) across all items across all time points was 0.52 but it was mostly >.65. The model yielded factor scores with determinacies for attention deficit symptoms and hyperactivity/impulsivity measured across time of > .98 with factor score variances ranging from 0.81 to 0.92 for attention deficit symptoms and from 0.75 to 0.91 for hyperactivity/impulsivity.

The analogous model for reactive and proactive aggression also fit well (CFI=.97, TLI=.97, RMSEA=.03, SRMR=.03) with within-wave factor correlations ranging from *r*=.65 to *r*=.70. The minimum covariance coverage was .50 but it was mostly >.65. The model yielded factor scores with determinacies of .95 and above for all factors. Factor score variances ranged from 0.67 to 0.82 for reactive aggression and from 0.33 to 0.71 for proactive aggression.

Univariate growth curves

Model fits for growth curves with linear and linear + quadratic growth are provided in Table 2. All models fit reasonably well by conventional criteria (e.g., Hu & Bentler, 1999). For attention deficit and reactive aggression, models including a quadratic growth factors were judged superior based on the ΔBIC >10 criterion. For hyperactivity/impulsivity and proactive aggression, models including only linear growth were judged superior. Parameter estimates for the best fitting growth curve models are provided in Table 3 and the mean growth curves plotted in Figure 1. These illustrate that all four phenotypes showed decreases over time on average.

Multivariate growth curves

In the context of other relevant equality constraints, the attention deficit and reactive aggression intercept and slope cross-phenotype correlations were 0.59 and 0.54 respectively. The corresponding values for proactive aggression were lower at 0.49 and 0.50 respectively. This difference in cross-phenotype correlations was statistically significant [χ^2 (2)=75.36, p<.001]. In the analogous hyperactivity/impulsivity model, the intercept and slope cross-correlations with reactive aggression were 0.70 and 0.65. The corresponding values for proactive aggression were 0.60 and 0.64. This difference in cross-phenotype correlations was statistically significant [χ^2 (2) = 76.11, p<.001].

Discussion

In this study we evaluated the hypothesis that there is a close developmental coupling between ADHD symptoms and reactive aggression. Our results broadly support this claim with moderately strong cross-phenotype correlations in the components of growth curves between ADHD symptoms and reactive aggression. We also hypothesised that the developmental coupling between ADHD symptoms and proactive aggression would be weaker than that of reactive aggression. Our results provide some support for this idea: the cross-phenotype correlations in the components of growth curves were always smaller when ADHD symptoms growth curves were paired with proactive aggression than when they were paired with reactive aggression.

The average growth curves for attention deficit and hyperactivity/impulsivity indicated that both phenotypes exhibit overall declines from age 7 through to age 15. This is consistent with previous studies suggesting that both the prevalence of ADHD and symptom levels within individuals decrease with age (e.g., Faraone et al., 2006; Monuteaux et al., 2010). In the case of attention deficit symptoms, there was some evidence that this decline was non-linear, even showing a possible increase towards later adolescence. Thus, hyperactivity/impulsivity symptoms showed a much more consistent and definitive decline than attention deficit symptoms. This is in line with past research suggesting that while a decline in hyperactivity/impulsivity is reasonably consistently observed, evidence for a decline in attention deficit symptoms is much more equivocal (e.g., Döpfner et al., 2015; Hart et al., 1995; Lahey, Pelham, Loney, Lee & Wilcutt, 2005). The explanation for these differences in trajectory may be in a differential dependence on specific and differentiable executive functions. For example, Miller et al. (2013) found that while individuals with ADHD who showed improvements on global executive function measures showed global ADHD symptom improvements, improvements in response inhibition were specifically related to improvements on hyperactivity/impulsivity. These developmental differences support the practice of making a distinction between attention deficit and hyperactivity/impulsivity in empirical research, even in spite of their strong cross-sectional correlation.

The average growth curves for reactive and proactive aggression suggested linear declines in both from age 7 to 15. The declines in reactive aggression were strongly and significantly correlated with declines in hyperactivity/impulsivity (linear slope correlation of r= .65) and to a lesser extent with attention deficit symptoms (linear slope correlations of r=.54). The declines in proactive aggression were also significantly associated with declines in hyperactivity/impulsivity (r=.64) and attention deficit symptoms (r=.50) but in both cases this was to a lesser extent than when paired with reactive aggression.

The differential associations between the developmental trajectories of reactive versus proactive aggression and ADHD symptoms supports our hypothesis that reactive aggression would show a particularly strong developmental coupling to ADHD symptoms as compared to proactive aggression. This hypothesis was developed from the observation that emotional impulsivity – a core feature of ADHD – also characterises much of reactive aggression. An important future direction will be to test this notion at the level of the putative underlying process, by examining whether emotional impulsivity and associated neurocognitive variables are developmentally coupled to ADHD and reactive aggression and explain their developmental association. Recent studies have also suggested finer distinctions within reactive aggression that may be relevant for this hypothesis. Smeets et al. (2016) factor analysed a set of proactive and reactive aggression items and found that a three-factor solution provided the best description of the data. The three factors could be characterised as proactive aggression, reactive aggression due to internal frustration and reactive aggression due to external provocation and it would be of interest to establish if these forms of reactive aggression are differentially related to ADHD symptoms, within- and between-individuals.

The differential relations of reactive and proactive aggression to ADHD symptom trajectories were found in spite of the fact that the two forms of aggression are highly correlated with one another developmentally (e.g. their intercept and slope covariances in the current study were around r=.87 and r=.95). Unfortunately, we did not have sufficient numbers of items to obtain

reliable measures of *unique* variability in proactive and reactive aggression such as might be obtained from a bi-factor measurement model with orthogonal general, reactive and proactive aggression factors (e.g. see Revelle et al., 2009; Murray & Johnson, 2013). Based on our hypothesis, we would expect that first residualizing on general aggression would yield stronger evidence of differential developmental coupling between ADHD symptoms and reactive versus proactive aggression. Indeed, in the current study though statistically significant, the magnitude of the difference in strength of developmental coupling between ADHD symptoms and reactive versus proactive aggression was relatively small.

Another limitation of the current study that could be addressed in future research is that we used a combined measure of hyperactivity/impulsivity symptoms where separate measures of hyperactivity and impulsivity would have provided a more fine-grained analyses. In particular, to the extent that the two dimensions could be distinguished from one another empirically, we would have predicted a stronger developmental coupling of the latter dimension with reactive aggression. Second, we used only a single informant to assess ADHD symptoms because only teacher-reported data was available in a comparable format across the entire age range from 7 to 15. It is common to find substantial inter-rater discrepancies on ratings of psychopathological behaviours in childhood and adolescence (e.g., Achenbach, 2005). This should be addressed in future research employing multiple raters such as peers, parent and self-reports, in addition to teacher reports. In particular, as there may be important contextual influences on the expression of ADHD symptoms (e.g., Rommelse, Bunte, Matthys, Anderon, Buitelaar & Wakschlag, 2015) it will be important to obtain ratings from individuals who observe the target in different environments (e.g., home versus school). Finally, ratings in the current study may have been affected by a 'halo effect': an inflation of inter-correlations because of a tendency to falsely ascribe symptoms to an individual who displays conceptually related symptoms (e.g., Hartung et al. 2010). They may also have been inflated by a common measurement method across all phenotypes; a limitation that could be addressed in future research using a multi-trait, multi-method or similar design (e.g., Podsakoff,

MacKenzie, & Podsakoff, 2012). However, it is unlikely that this would have affected some pairs of phenotypes more than others and by extension, the patterns of differential associations between ADHD symptoms and subtypes of aggression. As regards to our statistical approach, we note that modelling variation in individual trajectories as continuously distributed (as in bivariate latent growth curve modelling) acknowledges heterogeneity in trajectories; however, growth mixture approaches (modelling different subgroups defined by similar trajectories) may provide a complementary framework for summarising this heterogeneity in terms of meaningful subgroups. We, therefore, recommend that future studies also consider whether meaningful trajectory subgroups defined by combinations of ADHD symptoms and proactive and reactive aggression can be found. Finally, many of the arguments of the current study also apply to the impulsive behaviours observed in conduct disorder (CD) and oppositional defiant disorder (ODD), both also strongly correlated with ADHD symptoms (e.g. Falk et al., 2015). Therefore, a potentially interesting extension to the current study would be to conduct analogous tests of the developmental relations between the impulsive versus non-impulsive behaviours beyond aggression associated with CD and ODD.

Conclusions

Attention deficit and hyperactivity/impulsivity symptoms show strong and significant developmental relations with reactive aggression. To a lesser extent they also show developmental relations with proactive aggression. This is consistent with the idea that there is substantial overlap in the underlying (and developmentally maturing) neurocognitive architectures of hyperactivity/impulsivity and the reactive aggression subtype of aggression in particular. In this way, our results provide an important extension to the cross-sectional data showing differential relations between ADHD symptoms and reactive versus proactive aggression. These results suggests that children showing high levels of hyperactivity/impulsivity are at the greatest risk of exhibiting reactive aggression but that it is likely to improve and improve in tandem with hyperactivity/impulsivity symptoms over the course of development. Given the differential patterns

of developmental relations between attention deficit and hyperactivity/impulsivity and reactive and proactive aggression, out results also underline the benefits of making distinctions between sub-dimensions of both ADHD and aggression when aiming to illuminate developmental mechanisms.

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Table 1:

Attention deficit symptoms hyperactivity/impulsivity symptoms, proactive aggression and reactive aggression items

SBQ Subscale	Item content in English	Baseline mean (SD)
Attention deficit	He/She cannot settle to anything for more than a few moments.	2.23 (1.14)
Attention deficit	He/She is distractible, has trouble sticking to any activity.	2.50 (1.24)
Attention deficit	He/she can't concentrate, can't pay attention for long.	2.35 (1.17)
Attention deficit	Is inattentive.	2.27 (1.11)
Hyperactivity/impulsivity	He/She is impulsive, acts without thinking.	2.24 (1.17)
Hyperactivity/impulsivity	He/She hasdifficulty awaiting turn in games or groups.	2.28 (1.23)
Hyperactivity/impulsivity	He/She can't sit still, is restless, or hyperactive.	2.12 (1.20)
Hyperactivity/impulsivity	Fidgets.	1.99 (1.17)
Proactive aggression	He/She threatens people.	1.32 (0.66)
Proactive aggression	He/She encourages other children to pick on a particular child.	1.41 (0.74)
Proactive aggression	He/She tries to dominate other children.	1.62 (0.94)
Proactive aggression	He/She scares other children to get what he/she wanted.	1.29 (0.66)
Reactive aggression	He/She reacts in an aggressive manner when teased.	2.01 (1.07)
Reactive aggression	He/She reacts in an aggressive manner when contradicted.	1.72 (0.94)
Reactive aggression	He/She reacts in an aggressive manner when something is taken from him/her.	2.07 (1.08)

Table 2:

Model fits for linear and linear+ quadratic growth models

Model	Chi-square	*df*	CFI	TLI	RMSEA	SRMR	BIC	AIC
Attention deficit symptoms								
Linear	179.23	25	0.983	0.981	0.063	0.031	24624.954	24523.124
Linear+ Quadratic	130.077	21	0.988	0.984	0.058	0.027	24605.239	24481.971
Hyperactivity/impulsivity symptoms								
Linear	242.994	25	0.976	0.973	0.075	0.035	24359.748	24257.918
Linear+ quadratic	206.216	21	0.979	0.972	0.075	0.034	24352.407	24229.140
Reactive aggression								
Linear	242.381	25	0.966	0.961	0.074	0.043	26405.420	26303.59
Linear+ quadratic	201.688	21	0.971	0.962	0.074	0.037	26394.165	26270.897
Proactive aggression								
Linear	469.256	25	0.927	0.918	0.106	0.067	23858.029	23756.199
Linear+ quadratic	436.07	21	0.932	0.909	0.112	0.057	23854.281	23731.013

Table 3:

Key parameters from univariate growth curve models

	Attention deficit symptoms	Hyperactivity/ impulsivity symptoms	Reactive aggression	Proactive aggression
Intercept Mean	-0.036	-0.053	0.017	0.016
Intercept variance	0.709	0.630	0.329	0.397
Linear slope mean	-0.295	-0.333	-0.250	-0.372
Linear slope variance	2.512	0.628	1.507	0.428
Quadratic slope mean	0.239	**-**	-0.052	**-**
Quadratic slope variance	1.701	**-**	0.398	**-**

Figures

Figure 1: Mean growth curves for attention deficit symptoms, hyperactivity/impulsivity symptoms, reactive aggression and proactive aggression